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OSTEOPATHIC MEDICINE

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With this issue of OSTEOPATHIC MEDICINE we close volume II. We apologize for the delay, and for bringing the volume to a close by means of a combined November-December issue. We are compelled to do this by circumstances beyond our control.

To cooperate with the war effort, to conserve paper, and the time of the overburdened staff of the Hospital and College, volume III will be issued quarterly as of March, June, September, and December, 1945.

EDITORIAL BOARD.

THE PSYCHOLOGY OF THE CASUAL*

ROBERT E. BACHMAN, D.O.

It was my misfortune to be injured in training in this country and subsequently I spent an aggregate of twelve months in various Army hospitals. Unfortunately I am not a psychologist. I have, however, been a casual, so that I have the experience if not the ability necessary to report to you on the psychology of the casual.

Roughly, in Army lingo, a casual is a soldier without an assignment, and by far the majority of them are injured or wounded, and are hospitalized. At least these are the ones in whom we are interested in this discussion.

Questions which many of you may ask are, "Why the psychology of the casual?" "Why does this group of men fall into a separate psycho-

* Read on the General Program of the Graduate School, Philadelphia College of Osteopathy, Philadelphia, November 9, 1944.

logical category?" Well, let's examine the factors which operate to change the mental attitude of the casual.

Before his injury the average casual was a normal young man. He was adaptable to environmental changes and he took Army life in his stride. Oh yes, he griped and complained and didn't like it, but he accepted it as inevitable and he lived from day to day knowing that some time it would be over. Moreover he was busy, he was tired by the time the day was over, and he slept soundly. He had fun too. Here was something new for him. The bars had been let down. He went to town knowing he wouldn't meet anybody from home except possibly another soldier. So he drank a little more than he would have at home, he gambled, he whistled at the girls, and was a little like a mischievous youngster. But he wasn't bad, this average casual, he was just trying to prevent boredom and using the limited facilities available to him to do so.

He became injured and he was hospitalized. Then the picture changed somewhat.

First of all he was far away from home and no visitors came to see him. A few of his close buddies dropped in during the first week but then they forgot about him. He made friends of the other patients, true enough, but he didn't quite understand why his old friends let him down, and he thought of home more frequently now. He had time to think, too much time to think, and not much to guide his thoughts into proper channels. He read when the first pain was gone, light reading just to pass the time, but soon he lost interest and couldn't concentrate. He decided to read something deeper, maybe improve himself for the future, but that was a passing fancy. He was offered correspondence courses by the Army, high school or college. He enrolled, but gave it up at the end of a week.

Despair deepened during this period and here he saw an occasional ward mate fall into the abyss which the Army terms the psychoneurotic state. Possibly he wondered once or twice whether he was as sane as he had been. He wondered many times about the care he was receiving and discussed it freely with his neighbors. He didn't know if the care was good or poor, but he judged it wasn't good because it was impersonal. A few times a week the medical officer went through the ward and gave him a quick glance, usually without more than a word. He got the idea that they didn't give a damn if he recovered or not. In some cases he was right.

Every ten days or two weeks, six or eight doctors came through the ward. They discussed his case pro and con in front of him and argued. He didn't like that! He was another piece of equipment completely expendable, and he could be discarded without annoyance to the Army. All of this he sensed, and he definitely didn't like it. He had been a per-

sonality, and he was deeply perturbed about being treated in the abstract. In time our casual discovered that he had a permanent injury. His morale went down even lower, especially since many of his friends had been discharged during this time, gone back to the Army, and quickly recovered from any psychological wounds.

But our injured friend is improving, and finally he is told he can get out of bed. This is the first good news he has had for months. He is now able to see an occasional movie, and to use the Red Cross recreational room. Again his enthusiasm wanes. These things look rosier from a distance, and he soon spends most of his time in the ward. He wonders why he can't go home to convalesce, but of course he cannot, so he gripes about it, and curses the powers that be. If he gets a short week-end pass to town he is likely to spend it in drinking, for unfortunately, he has learned that drinking brings forgetfulness. He plays cards, or rolls dice for a week or two after payday, and he plays from morning till night—until he is without funds. Then he wanders around aimlessly with time hanging very heavy on his hands until next payday.

His biggest thrill now is to talk to his buddies about home and friends. He listens to their stories, and nostalgia hangs in the air like a cloud, but during these sessions he is happy. Usually he makes one trip a day to the PX. He only buys a soda and a magazine, and window shops, but it's one of the highlights of the day.

I have tried to show you the factors affecting the psychology of the injured service man. I think you can agree with me that the factors tend toward an unhealthy attitude, a morbid, beaten, mental state. The soldier injured, out of combat, feels like a weight around the neck of his country. He is not a shouting patriot, but he would have liked to have done his duty. Instead of which he has added nothing, and has been a burden to the army. He fully realizes these facts and he is angry with himself for being injured but he transfers that anger to other channels. He becomes bitter toward the Army, the governmental system, and many other things. Most likely this type of person will terminate his hospital stay by being discharged from the Army. I believe he will adjust himself in time to civilian life, and I don't anticipate any great problem from his type. You will see him frequently in your offices and we shall meet him again a little later.

Now for the battle casual, the man injured in actual combat. It may seem surprising that there should be any great difference between these two types of casuals, and yet in my experience there is a considerable gulf between the mental change that occurs in the two cases. The reasons are not hard to understand. The man who has received wounds in battle is justifiably aware that he has done his duty. He was called on to risk his life, and he has done so for his country. He is by every standard a hero. He has in most cases few regrets and, unless his injury is extremely

disabling, he looks forward eagerly to the future. His head is held high. His morale is boosted by other factors. He is back in this country and, from witnessing the reactions of others, I think this must be a great feeling. The Army policy is usually to send battle casualties to hospitals near their homes when they return from overseas. So this type of casual soon sees his family and friends and, as soon as he is able, he is permitted short leaves from the hospital. He is treated with considerably more deference by the hospital personnel and his complaints receive immediate attention. He is called on to relate his battle experience to the other patients, and he soon becomes *somebody* in his own ward. This factor would not operate where battle casualties made up all or most of the hospital census.

From our standpoint it would be valuable to understand how the casual views his injuries. Or, rather, how he will view them in civilian life. I think you will be pleasantly surprised in the future when you meet the former casual. He will make every attempt to minimize and overcome his handicaps. He wants nothing but a chance to earn his living as he did before. He has dreamed of home and the old ways of life so long, that he will gladly put forth every effort to make that dream a reality.

Understand that the pictures I have tried to portray of the casual are the result of much averaging of emotions and personalities. These men came into the service from every walk of life and with every possible mixture of mental characteristics. They were subjected to a rather uniform environment and they reacted in every conceivable way. For example, I have seen men definitely benefit from their hospital stay. Some have learned much that is new to them, or supplemented learning in their particular fields. Others have become acquainted with music or other of the fine arts, and their lives have been greatly enriched. On the whole, though, the tendency from a psychological standpoint has been downward. There will be many more casuals who have to unlearn mental habits acquired in the hospital, than the number who will have benefited from their stay. But I believe most of these who have acquired warped mental habits will give them up when they are civilians again, and that relatively few will carry mental scars.

As physicians, you want to know what the doctor-patient relationship with these men will be. Here again it will vary widely but I am sure of several facts.

The casual will be delighted to be a private patient. He has become used to the impersonal care of the Army. To have his own doctor with whom he can discuss his case openly will be a great advantage to both parties. He will place his confidence in you wholly, if you justify it at all, and he won't expect miracles because he hasn't seen them. He has seen on the average only fair results with unusually long convalescent periods. I must state here, in defense of the Medical Corps, that men

are kept in Army hospitals until they are considered fit for active duty, a fact which causes unusually long periods of hospitalization. But these things have not made him sceptical of doctors; on the contrary, he is anxious to get out and choose his own course of treatment. That choice will be, I am sure, away from any socialized form of treatment. I have heard, repeatedly, remarks against socialized medicine by men who previously had favored or, at least, had not frowned upon it. Remember, however, that this sentiment is that of the casual, and his voice may not be strongly heard because he will be in the minority of the returning servicemen when the war is over. No, I don't think you will have any trouble with the casual. He'll give you a fair trial and he'll cooperate fully. The rest is up to you.

As for correcting his psychology: In most instances that will be done automatically by living a normal life again. The things which in my mind would cause trouble, would be a depression, or some personal problem brought about by his absence. In either of these events, the anger and the bitterness of the casual might again flare up, and he might become a difficult patient and a poor citizen. In other words, if he can assume a life closely paralleling his old civilian days, well and good; if not, his mental attitude will become worse and worse. As his doctor, my advice to you is to treat him as though he had never been away, as just another neighbor whom you respect and are glad to serve.

Now a word about the attitude of the public toward the injured service man. You've heard this before, but it will bear repeating because you are the logical persons to educate the general public in these matters. The injured service man doesn't want to be reminded of his disability. He is trying to forget it. A casual question as to his progress may be in order, but don't dwell on it. Never stare at his injury or even allow him to know that you are aware of it. He will not appreciate any help in doing routine tasks. Remember that most of the time he must be on his own and he would like to get used to it. Don't warn him to be careful. He knows his capabilities and has to be the judge of what he can and can't do.

There will be, resulting from this war, a great number of men who are purely psychological casuals. They have received no physical injury, or at least it played a minor role in their present complaint. They are the men who could not adapt themselves to Army routine or discipline, or they have been unable to withstand the shock of battle. Of the former group, I have seen many and they range in severity from almost unnoticeable symptoms to violent degrees of insanity.

In tracing past histories with some of these patients, I would conclude that they all showed neurotic tendencies prior to service in the Army. An already faltering nervous system was just unable to cope with the complete change required by Army service. Neurotic tendencies quickly became exaggerated, and in some cases reached the point of in-

sanity. I would assume that severance from the Army in most of these cases will be followed by a return to the patients pre-war status. The true insanities were probably headed for institutions long ago and, unfortunately, their trip was speeded by their Army service. I know nothing of the effects of combat on the nervous system but the victims of this condition will present a problem to us all in the future. I hope the solution will soon be available to us all.

I have tried to cover the various types of casuals and broadly what you may expect of them and how you may best handle them. Now I'd like to say a bit about the contribution of osteopathy to these men.

First, will you see them in any appreciable numbers? Well, I think you will. It has been amazing and gratifying to me to hear over and over again the wish for osteopathic care. I hardly believed that that many persons were familiar with osteopathic physicians. I don't believe keeping us out of the Army as a profession has hurt our prestige at all. In fact, it probably has enhanced it with the casual.

To illustrate this point I would like to relate some of my own experiences. I hope you will understand that any of you might have done the same or better work. When I was able to walk I was transferred to a convalescent hospital which had just been opened. It was the policy of this institution to hold group discussions on world affairs twice weekly. On one afternoon during our discussion the commanding officer of the unit had visitors from Washington to inspect the unit and he brought them to our meeting. When they arrived I was in the midst of expounding my favorite post-war theme. Apparently the Colonel agreed with my views for he stopped me in the corridor later and thanked me for a good discussion. He asked me what my civilian occupation had been and when I told him, he requested me to train a few men for a physiotherapy unit which had just been opened in the hospital. I agreed and went to work the following day.

It is the Army habit to commission gym teachers, or other personnel with even less background, as physiotherapists. Because of the shortage of trained persons, two privates were assigned to me for instruction. Our patients all came from the main hospital where they were receiving physiotherapy daily. It consisted mainly of baking the injured part and very gentle massage. It was rather smirked on by the patients and they went because they had to.

In our unit the care was somewhat osteopathic since I knew very little about physiotherapy. In a short time the boys not only came willingly but actually tried to be placed on the physiotherapy list. They almost fought for places at my section of the room. Not because they liked me, but because they quickly discovered that they were improving. The word sped rapidly around the hospital and soon patients were stopping

me in the hall for information or to request treatment. The Captain who had nominal charge of the physiotherapy room didn't like me at all, but because the Colonel had made the assignment, he had to put up with me. I had requests from officers for treatment for themselves and for their families. The patients tried to consult with me rather than go on sick call, a practice which I tried hard to break up. Some of the results obtained were excellent and I have the gratitude of many of those men. And all this because first, I knew something about manipulative therapy and secondly, I did my best for every case under my care. I showed a little interest, and really tried to help them.

Yes, you'll see these casuals, and the same combination of manipulative care and real personal interest will bring them to you in numbers. They will probably get some treatment through the Veterans Administration but they will discover that it is the same impersonal treatment they disliked in the Army. Consequently, they will look for help elsewhere. Many of them will come to you because they have been dissatisfied with medical treatment. They will be good patients. Just meet them halfway and do your best for them. They deserve that.

CAUDAL MANIKIN

JULIAN LANSING MINES, III
Associate in Obstetrics

The use of continuous caudal analgesia in obstetrics is now approaching its maturity. This method of obstetrical analgesia, as laid down by Drs. Robert Hingson and Waldo Edwards, has proven itself to be the most sensational addition to the armamentarium of the obstetrician during the past decade.

We, at the Osteopathic Hospital of Philadelphia, have been doing extensive work in this field during the past year.

When a procedure such as this is devised, there immediately arises the problem of properly training men in its use. Unfortunately, many hours of training and study are necessary to master the technique needed to carry this procedure through its many ramifications successfully.

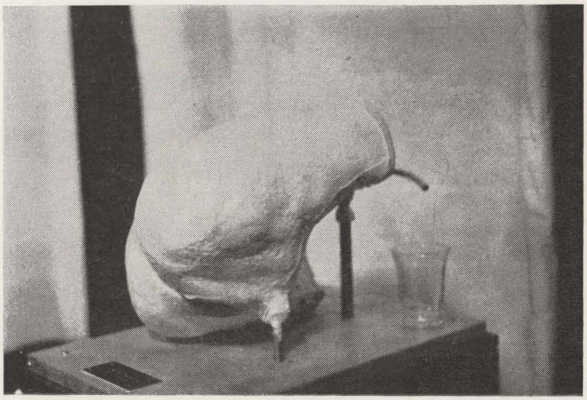
Because of possible permanent harm that could be done to the patient by the novice, we have devised a manikin to be used in teaching the proper method of needle insertion.

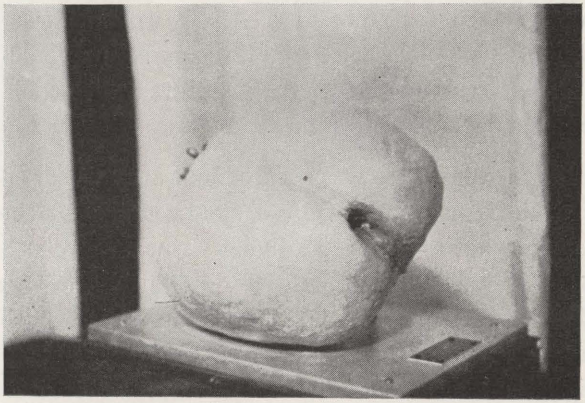
After much experimentation, a pseudo-pelvis, complete with simulated bones, ligaments, muscles, and skin has been constructed including a caudal hiatus and canal, as well as a movable coccyx. The bony structures are of metal, exactly duplicating a human pelvis. This skeletal structure is covered with rubber of varying consistencies to simulate muscular and ligamentous tissue, and the entire form is covered with a synthetic material having many of the characteristics of skin. The manikin is colored to resemble the normal deposition of body pigmentations.

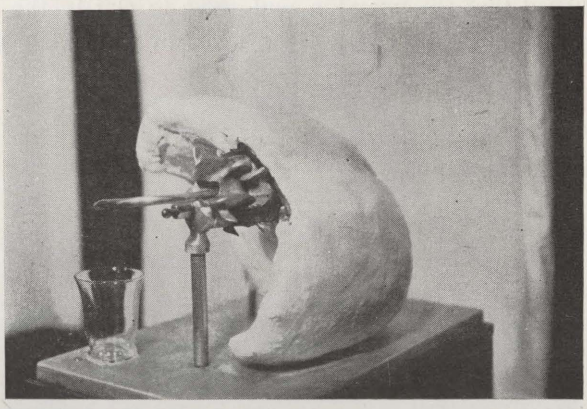
A complete vulva, vagina, and rectum are incorporated so that natural landmarks can be utilized.

A tube is inserted at the proximal end of the caudal canal, and made to protrude from the upper end of the manikin. Upon correct insertion of the needle, the injected fluid will flow through the tube into a glass receptacle.

The accompanying photographs were taken in the Philadelphia College of Osteopathy.







THE FEMALE PERINEUM

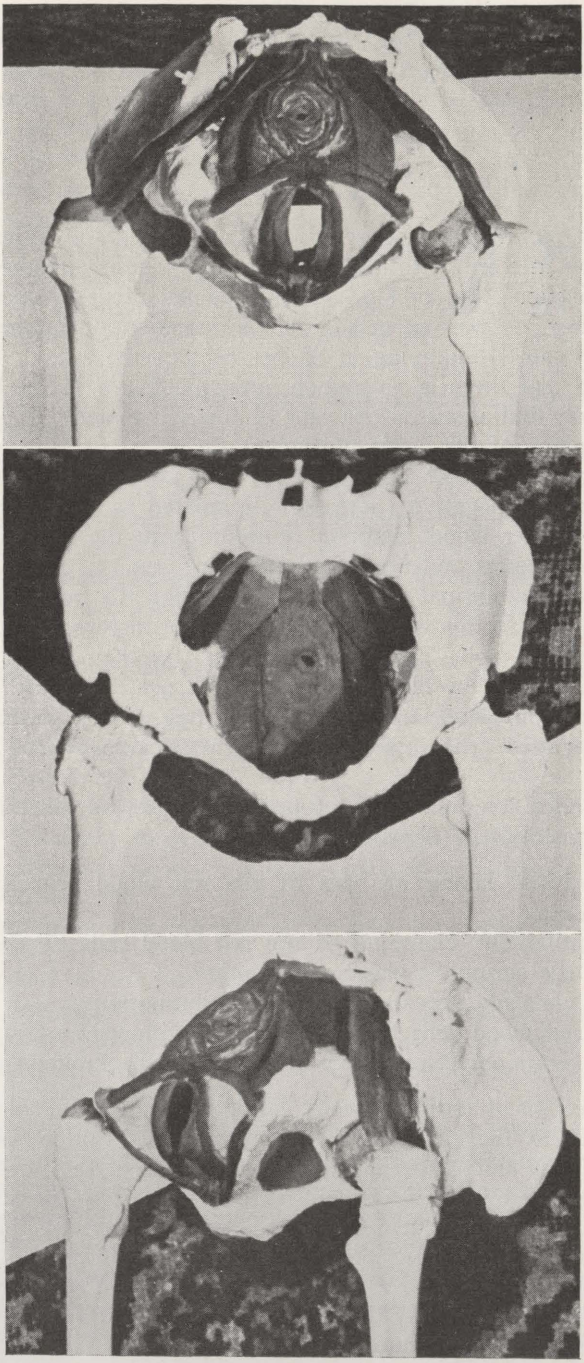
JULIAN LANSING MINES, III
Associate in Obstetrics

For the instructor the problem of teaching the anatomy of the female perineum has always been difficult. For many medical students the problem of understanding and visualizing this complex structure has been almost insurmountable. The difficulty of the problem would seem to be that of forming a mental picture of a closely woven, three dimensional structure. The best possible solution of the problem would seem to be the use of a lifelike reproduction of the structure which could be handled by the students, and examined from all angles.

A suitable gynecoid pelvis was secured and articulated, and the upper one-third of both femora attached. After considerable search for a material having properties similar to living tissue, a special "Latex" was selected as most suitable because of its durability and great elasticity. With it all the muscles of the female perineum, and the pyriformis muscles, are represented in natural color. Each muscle is attached to its appropriate origin and insertion, and each indicates the direction of the muscle fibers found in the corresponding living muscle.

It is believed that this manikin will serve to clarify the anatomy of the female perineum, and so simplify a problem which has always been difficult for both teacher and student.

The accompanying illustrations were made from photographs taken at the Philadelphia College of Osteopathy.



THE Rh FACTOR AND ITS RELATION TO THE PRACTICE OF OBSTETRICS

JULIAN LANSING MINES, III
Associate in Obstetrics

The Rh factor was discovered by Landsteiner and Wiener in 1940. Rabbits were injected with the blood of the rhesus monkey. After a series of injections, the rabbits' serum clumped the red blood corpuscles of the monkey, as was expected. Upon injection of human blood, the rabbit serum caused clumping in 85 per cent of the cases. Therefore it was assumed that there is a common antigenic factor in the blood of the rhesus monkey and about 85 per cent of the white race. It is found in a larger percentage of the black and yellow races.

The percentage which have the Rh factor are called Rh positive. The remainder are Rh negative (hereafter designated as rh). The Rh is independent of other blood factors. It occurs with the same frequency in persons of all groups and types, regardless of sex.

There are no normal agglutinins to combat Rh in the Rh negative (rh) individual. In this way it differs from the factors A and B. Therefore, Rh negative (rh) persons do not have in their serum agglutinins capable of clumping the blood of Rh positive persons, which may be introduced into their blood stream. However, agglutinins are manufactured in the Rh negative (rh) individual after the introduction of Rh positive blood.

For a fuller comprehension of the Rh factor, we must review some of the fundamentals of hematology.

1. Hemolysis—laking or lysis of the red blood cells by serum or plasma.
2. Agglutination—clumping of the red blood cells of one individual by the serum or plasma of another.
3. Two elements necessary for hemagglutination—
   a. red blood cells be clumped (agglutinogen)
   b. serum or plasma which brings about the clumping (agglutinin)

The injection of red blood cells (antigen) of one animal species into another leads to the production of agglutinating antibodies or hemagglutinins. They clump specifically the blood cells of the injected species. The agglutination of red blood cells of one species by the serum of another is not surprising, but when Landsteiner discovered the agglutination of red blood cells of one person by the serum of another, both being normal, healthy individuals, a new problem arose. From this discovery blood typing originated. With the discovery of the Rh factor, further reclassification of blood became necessary.

For many years certain pathological conditions were found in the newborn: erythroblastosis foetalis, foetal hydrops, and icterus gravis. These were classified individually, all having vague etiologies. Erythroblastosis foetalis is primarily an acute hemolytic anemia of the newborn, probably due to an agglutinative hemolytic process. Recent studies of the Rh factor have given substantial proof of this fact. One of the important results of these experiments is the grouping of several pathological findings in the newborn under one heading.

All of these conditions may exist separately or combined in a mild form and still be considered within the bounds of normalcy. When, however, symptoms are markedly distinct, a state of acute hemolytic anemia exists. For these symptoms to be present in the child, it is now known that the father *must* be Rh positive and the mother *must* be Rh negative (rh). Since Rh is a dominant factor, this combination produces an Rh positive offspring.

It is assumed that erythroblastosis foetalis results from the passage of Rh positive cells in the foetal blood via some placental mechanism into the maternal blood stream. There immunogens or agglutinins are formed in the maternal blood serum. These, in turn, filter back into the foetal circulatory system and cause hemolysis of the infant's blood cells. *Here we might say the baby receives a transfusion of incompatible blood from its mother.*

The results of this hemolysis are as follows:

1. Interference with serum protein metabolism.
2. Interference with liver metabolism.
3. Jaundice.
4. Renal failure—hydrops, etc.
5. Tendency to generalized hemorrhagic diathesis.
6. Presence of erythroblasts—nucleated, immature red blood cells.

(An attempt on the part of the baby to combat the anemia.)

Direct proof of the hemolytic nature of the disease can only be determined by the quantitative demonstration of derivatives resulting from the breakdown of hemoglobin, i.e., bilirubin and its products. Fecal bilirubin determinations are fairly accurate in estimating the amount of hemolysis present.

Fortunately, the first born seems to escape this phenomenon, apparently because the maternal immunogens developed during this pre-natal period are insufficient in number to give the maternal serum a high enough titre to cause acute hemolytic anemia in the child.

Subsequent pregnancies, however, will not escape some manifestations of erythroblastosis foetalis, ranging from a living child showing some or all of the above symptoms, to a premature stillborn or early abortion. This, presumably, is due to the concentration of immunogens in the maternal serum.

Suggested Treatment

If, through Rh factor determination in early pregnancy, the possibility of an acute hemolytic anemia in the infant is expected, early Caesarean section is suggested after the period of viability has been reached. It is thought that the greatest damage is done to the foetal blood stream during the last trimester. Following delivery these procedures in the management of the infant are recommended:

1. Vitamin K to shorten the blood coagulation time. (5 mgm. "Synkovite" daily.)
2. Dextrose in some form to support liver function.
3. Blood plasma to augment serum protein which is lost in the presence of edema (25-50 cc. daily).
4. *Whole blood that is Rh negative (rh) taken from a donor of the same general blood type who has never been transfused or given birth.*

   The blood of the child's father is of no value because it is Rh positive and will also be hemolyzed by the maternal immunogens circulating in the infant's blood stream. The blood of the child's mother is useless, even though it is Rh negative (rh), because it contains the immunogens manufactured to combat the baby's blood. If 75 cc. of the proper blood is given on 3 consecutive days, the replacement should be sufficient to show a satisfactory improvement in the blood picture.
5. The child should not be permitted to nurse from the mother's breast, as it has been proven that breast milk contains a relatively high concentration of immunogens.

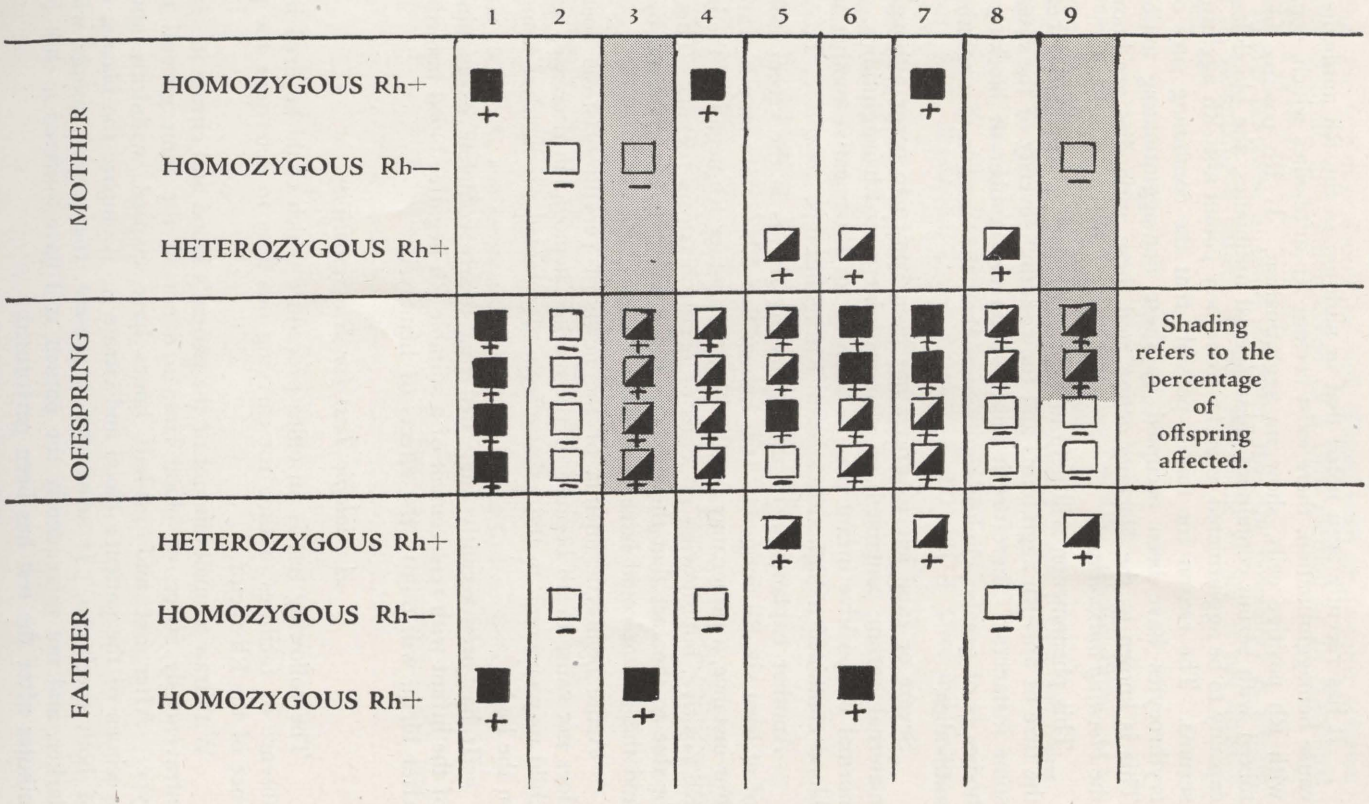
Homozygous and Heterozygous Types

The Rh factor of a person is the result of inheritance. He may inherit the Rh positive property from both parents, making him an RhRh, and therefore, homozygous. Likewise he may inherit the Rh negative (rh) property from both parents, making him an rhrh (homozygous).

However, he may inherit the Rh negative property from his paternal side and the Rh positive from his maternal side (see column 4 on chart) and become a heterozygous Rh positive, Rhrh, immune to the possibility of acute hemolytic anemia, due to the Rh factor. The only dangerous combination is that of an Rh negative (rh) mother and an homozygous or heterozygous Rh positive father (see columns 3 and 9 on chart), which will produce all or some offspring subject to erythroblastosis foetalis due to the Rh factor.

It should be borne in mind that any woman bearing a child should not receive a blood transfusion without first having an Rh determination made on her blood. If she is an Rh negative individual, she may be building up immunogens which would hemolyze any Rh positive blood she might receive.

INHERITANCE OF THE Rh FACTOR



Recent Rh Factor Studies

It has recently been noted that in addition to the Rh antibodies that cause hemagglutination, there exist certain Rh antibodies which, combined with Rh positive cells, show *no* agglutination. If Rh positive blood is mixed with serum containing these special antibodies, the blood loses its capacity to be agglutinated, even by extremely potent anti-Rh agglutinating serums. The reason for this is possibly that the combining sites on the erythrocytes have been occupied by these non-agglutinating antibodies. This is known as the *blocking action*, and these antibodies are known as the *blocking antibodies.*

This phenomenon will help to explain the lack of correlation between the titre of anti-Rh agglutinins and the resulting severity of the disease in some instances. Therefore it appears that the number of blocking antibodies is of greater clinical significance than the number of agglutinating antibodies.

Severe or fatal icterus gravis has been known to occur although the maternal serum contained no demonstrable anti-Rh agglutinins, or a normal Rh positive infant may be born of an Rh negative mother whose blood contained a high titre of anti-Rh agglutinins.

Another explanation of these puzzling results is the recent isolation of at least six Rh antigens. It is known that Rh factors may be variable. For instance, a father may be Rh_1 Rh_2, the mother Rh negative; the infant Rh positive, but showing no signs of erythroblastosis foetalis. This may be due to the fact that the mother's blood can only be sensitized by Rh_1 and this infant could have been Rh_2.

Acute hemolytic anemia of the newborn (erythroblastosis foetalis) does not cause death because of the marked hemoglobin decrease, but the child may succumb to the lethal effects of retained products of hemolysis in the body.

It has been recently suggested that nearly complete exsanguination of the infant with replacement of a suitable Rh negative blood immediately after birth, will nullify the effects of hemolysis.

A Biologic Test for Rh Sensitivity

The following test is an office procedure which could be used in the absence of facilities or time for carrying out tests to determine the presence of the Rh factor.

Withdraw a small amount of the patient's blood and citrate it. Inject intravenously 50 cc. of blood from a donor of the same general blood type. After one and one-half hours have elapsed, withdraw another specimen of the patient's blood and citrate it. Compare the plasma color of both specimens. If sensitivity is present, the second sample will be darker, and not infrequently the patient will have suffered a chill 50-60 minutes after the test has been performed.

Due to the fact that severe reactions may sometimes occur, it is believed that the test dose should be well diluted with an isotonic solution of sodium chloride, and administered by a drip method or in fractional doses over a period of 1 to 2 hours.

It should be borne in mind, however, that the possibility is always present of sensitizing a patient by this test if he or she is not already sensitized.

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ENDOCRINOLOGY IN OBSTETRICS

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The purpose of this article is to present a compilation of endocrine data which may be helpful in solving some of the problems confronting the physician practicing obstetrics.

Knowledge concerning the glands of internal secretion is far from being complete today, but the physician should have an up-to-the-minute comprehension of this system in order to offer proper scientific treatment methods to his patients. In the field of obstetrics, the endocrine system plays an important role, and a working knowledge of some of its intricacies is important to the obstetrician.

Endocrines During the Menstrual Cycle

As a review, the endocrine balances during the course of the menstrual cycle should be clearly understood. Because we must start somewhere, we shall take the anterior lobe of the pituitary gland as our keystone. Chart A is constructed in five tiers or sections, numbered from above downward, to show the interrelation of hormonal and biological changes, and the relation of all the changes to time during a menstrual cycle.

The two substances with which we will deal first are called Follicular Stimulating Hormone (F.S.H.) or prolan A, and Luteinizing Hormone (L.H.) or prolan B, indicated in section I. We will assume that during the first fourteen days of a twenty-eight day menstrual cycle F.S.H. is secreted. Let us suppose that ovulation occurs on the fourteenth day of this cycle, and from this day on till the twenty-eighth day, L.H. is the substance secreted by the anterior lobe of the pituitary gland. It has been recently postulated that the sudden changeover from F.S.H. secretion to L.H. secretion creates the impetus necessary for ovulation to take place.

Now examine section II of the chart, which indicates some of the changes taking place within the ovary during this twenty-eight day period. The Graafian follicle containing its ovum is maturing and migrating toward the surface of the ovary during the first fourteen days. It should be noted that of the thousands of follicles present in the ovary at the time of birth, less than four hundred ever reach maturity, migrate to the ovarian surface, and discharge their ova. Probably a reason for this is that many immature follicles must secrete estrogen in order that one may reach maturity. When this duty has been performed, these immature follicles atrophy. The follicle selected to mature enlarges as it migrates toward the ovarian surface, and in so doing produces within its cavity increasing

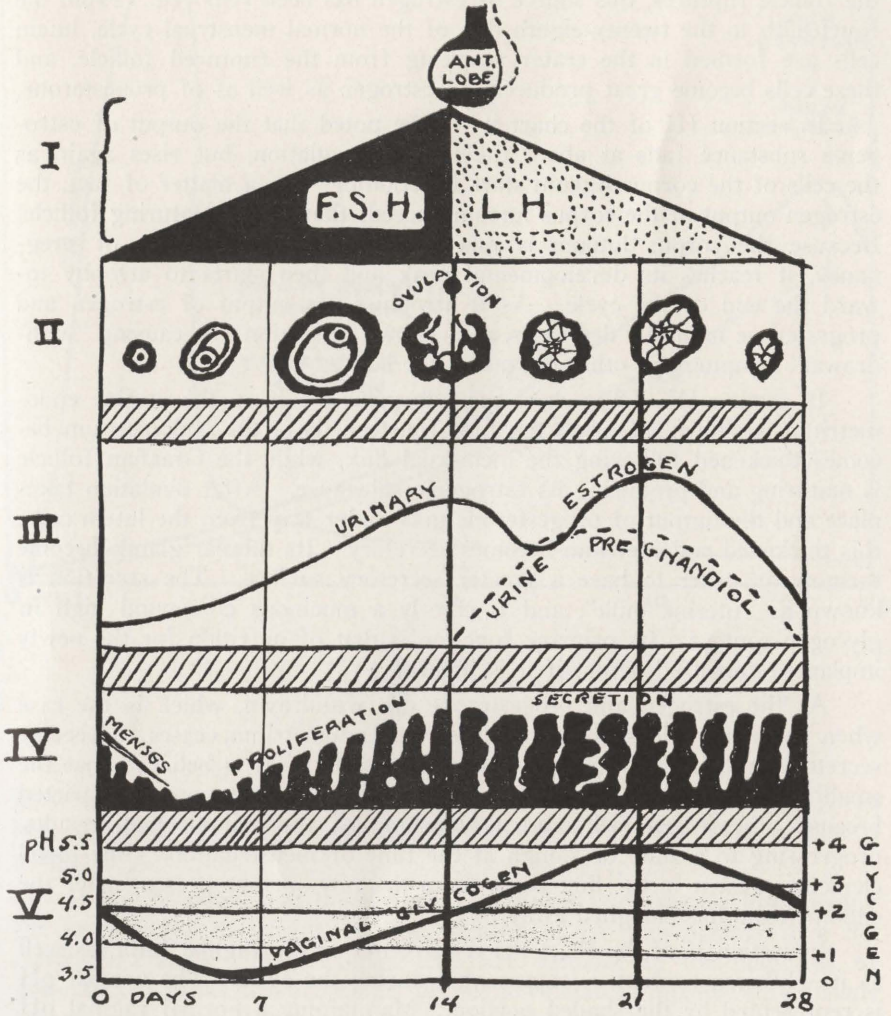
ANT. LOBE
I
F.S.H.
L.H.
II
OVULATION
III
URINARY ESTROGEN
URINE PREGNANDIOL
IV
MENSES
PROLIFERATION
SECRETION
V
pH 5.5
5.0
4.5
4.0
3.5
VAGINAL GLYCOGEN
+4
+3
+2
+1
0
GLYCOGEN
0 DAYS
7
14
21
28

CHART A

amounts of follicular fluid, high in estrogen content. Therefore, it is assumed that until the fourteenth day, the ovary is producing relatively large amounts of estrogenic substance. When ovulation takes place, and the follicle ruptures, this source of estrogen has been removed. From the fourteenth to the twenty-eighth day of the normal menstrual cycle, lutein cells are formed in the crater resulting from the ruptured follicle, and these cells become great producers of estrogen as well as of progesterone.

In section III of the chart it will be noted that the output of estrogenic substance falls at about the time of ovulation, but rises again as the cells of the corpus luteum start functioning. As a matter of fact, the estrogen output of the corpus luteum exceeds that of the maturing follicle. Because the corpus luteum is one of menstruation and not of pregnancy, it reaches its developmental peak and then starts to atrophy toward the end of the cycle. As it atrophies, its output of estrogen and progesterone naturally decreases, as is shown in section III, causing "withdrawal" symptoms in other organs of the body.

In section IV of the chart the estrogenic effect on the uterine endometrium is shown. During the first fourteen days, the endometrium becomes thickened following the menstrual flux, while the Graafian follicle is maturing and producing its estrogenic substance. After ovulation takes place and the output of progesterone gets under way from the lutein cells, this thickened endometrium becomes secretory. Its tubular glands become tortuous in order to have a greater secretory surface. The secretion is known as "uterine milk", and is chiefly a mucinous compound high in glycogen content. Its primary function is that of nutrition for the newly implanted fertilized ovum, if it is present.

As the estrogen and progesterone are withdrawn, which is the case when conception does not take place, the endometrium ceases to become secretory, and its blood supply becomes impaired. It is believed that the small arterioles which supply the basal endometrium become constricted because of estrogen lack, and a blanching of the endometrium results, progressing to a state of slough at the time of menstruation. The blood elements present in the flux come from the raw surface present after the endometrium has sloughed away.

In section V of the chart the relative pH of the vaginal vault, as well as its glycogen content, is represented during the cycle. The normal pH is represented by the shaded portion. Maintaining a normal vaginal pH by the use of estrogens, acidifying douches, and glycogen suppositories may be necessary to produce a normal bacterial flora and keep the vaginal epithelium normal.

Endocrines During Pregnancy

The foregoing statement of hormonal and associated changes occurring during a typical menstrual cycle has been reviewed to furnish a background against which the liberation of prolan, estrogen, and pro-

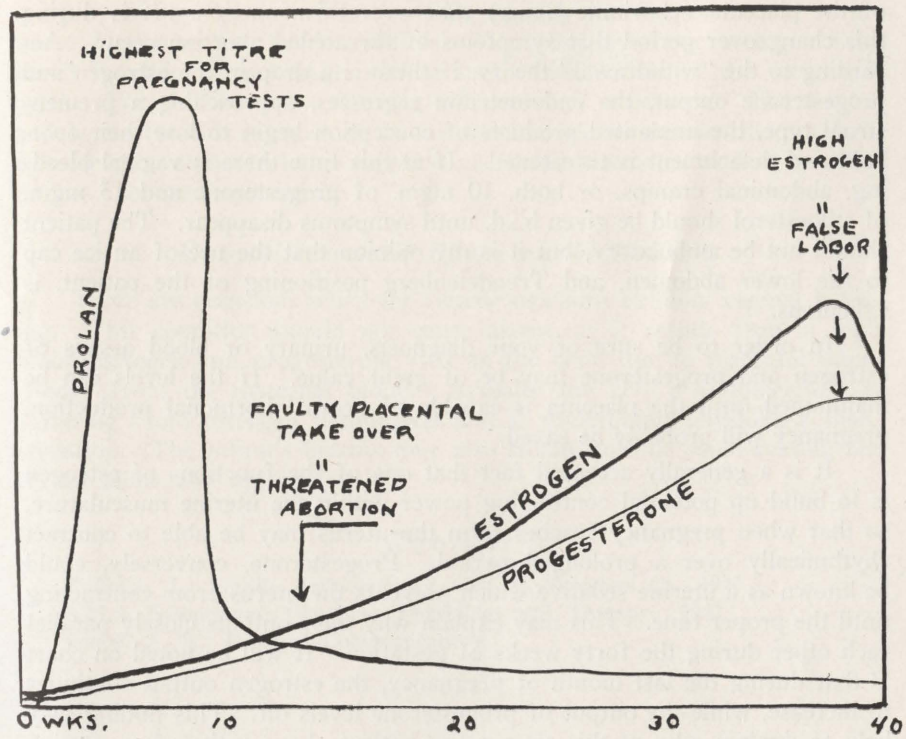

CHART B

gesterone during pregnancy may be projected and understood. These are indicated on chart B, which is subdivided into the forty weeks of pregnancy.

It will be noted that the secretion of prolan is extremely high during the early weeks of pregnancy, and because of its high urinary titre the Asscheim-Zondek and Friedman tests are valuable in the diagnosis of pregnancy. When the urine of a pregnant woman is injected into female rats or rabbits which have never ovulated, the high prolan content stimulates ovarian changes similar to those indicated in section II of chart A.

The urinary output of estrogen and progesterone is low early in pregnacy, becomes increasingly high as pregnancy proceeds, and it is important to note that their curves normally run rather parallel courses.

Near the fourteenth week of pregnancy, the corpus luteum, which has been the main source of estrogen and progestrone, is no longer able to produce sufficient amounts of these substances and the interstitial cells

of the placenta (chorionic tissue) take over this function. It is during this changeover period that symptoms of threatened abortion occur. According to the "withdrawal" theory, if there is a drop in the estrogen and progesterone output, the endometrium regresses, approaching a premenstrual type, the implanted products of conception begin to lose their foothold and detachment is threatened. If at this time there is vaginal bleeding, abdominal cramps, or both, 10 mgm. of progesterone and 15 mgm. of stilbesterol should be given b.i.d. until symptoms disappear. The patient should not be ambulatory, but it is my opinion that the use of an ice cap to the lower abdomen, and Trendelenberg positioning of the patient, is ridiculous.

In order to be sure of your diagnosis, urinary or blood assays of estrogen and progesterone may be of great value. If the levels can be maintained until the placenta is capable of normal hormonal production, pregnancy will probably be saved.

It is a generally accepted fact that one of the functions of estrogen is to build up potential contracting power within the uterine musculature, so that when pregnancy reaches term the uterus may be able to contract rhythmically over a prolonged period. Progesterone, conversely, could be known as a uterine sedative which prevents the uterus from contracting until the proper time. This may explain why their outputs closely parallel each other during the forty weeks of gestation. It will be noted on chart B that during the last month of pregnancy, the estrogen output continues to increase, while the output of progesterone levels off. This finding may help to explain why at this time many patients have rather firm uterine contractions and are said to be in "false labor."

The production of estrogen and progesterone in increasing quantities during pregnancy also causes the mammary glands to develop to the point where they will be capable of lactation when the proper time arrives. At the same time, "prolactin", the stimulating factor to lactation, is dammed up due to the presence of estrogen and progesterone in the blood stream. When the third stage of labor is completed, and the production of progesterone and estrogen is cut off, the "prolactin" is liberated and causes the normal mammary tissue to secrete. This should make the use of 100 I.U. of "prolactin" given b.i.d. for five days seem logical in the treatment of cases of insufficient lactation.

Urinary and blood estrogen and progesterone determinations are of diagnostic value in the case of suspected hydatiform mole and chorionic epitheliomas. If, after the placenta has been expelled, the levels remain high, chorionic neoplasms must be suspected.

Principles Underlying Suppression of Lactation

There are times when the physician wishes to "dry up" the breasts. By giving the patient 5 mgm. of stilbesterol t.i.d. for the first few days, and then tapering off the dosage over a two week period, a partial dam-

ming up of the "prolactin" may result, causing the suppression of lactation. However, the sudden withdrawal of this synthetic estrogen may result in undue hemorrhage, similar to the mechanism of the menstrual cycle. For this reason a tapering off period is necessary. The use of androgens for this purpose is, in my opinion, rather unscientific, as their sole function seems to be to suppress all female hormonal activity, a more or less "shotgun" procedure.

Maternal Hormonal Effects on the Newborn

There are occasions when the female newborn exhibits vaginal bleeding. This condition should not cause alarm, as it results from a high blood estrogen and progesterone titre in the child due to placental interchange from the maternal blood. Actually the child's endometrium is suffering from estrogen withdrawal and is undergoing physiologic menstruation. The infant's breasts may also lactate for the same reason, but if left alone will regress.

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DEATH BY CORONARY OCCLUSIVE DISEASE IN CARCINOMA OF THE PROSTATE: AUTOPSY REPORT OF A CASE

OTTERBEIN DRESSLER
Professor of Pathology
and
NORMAN ARENDS
Fellow in Pathology

Autopsy No. 44-359
Died: 10-24-44— 6:30 p.m.
Autopsy: 10-25-44—10:00 a.m.

Clinical Data

An elderly male patient was admitted to the Osteopathic Hospital of Philadelphia on October 21, 1944, with the chief complaint of shortness of breath. He also complained of pain and burning on urination. The dyspnea had begun 5 or 6 weeks prior to admission and, with it, tightness over the precordium. Dysuria had begun nearly 5 years before admission. He was aware that he was taking digitalis and was being treated for prostatism. His past personal history was insignificant except for scarlet fever in childhood.

Physical examination revealed an elderly male 75 years of age, weighing 145 pounds and being 5'4" in height. The pulse was 88 per minute, respirations 30 per minute, and blood pressure was 120 systolic and 70 diastolic. The myocardial tonus was described as poor with extra systoles occurring every 15-20 beats. The heart was enlarged, the apex impulse being found at the 6th left interspace, 12 cm. to the left of midline. Pitting edema was present distal to the knees. The liver was enlarged, extending 2 fingers below the costal arch. Breath sounds were absent in the bases of the lungs.

Examination by laboratory methods revealed a few hyaline and granular casts in the urine with a 1 plus albumin. The blood count showed a white count of 14,600 composed of 10 per cent lymphocytes, and 89 per cent neutrophiles (1 per cent mononuclears completed the count) with a multiple index of 4.0. Erythrocyte and hemoglobin levels were average for the individual's age and sex. Blood sedimentation showed an index or fall of 3 mm. in one hour. A blood urea nitrogen level was found to be 31.2 mgm./100 cc. An electrocardiogram gave the interpretation of a right bundle branch block with anterior coronary infarction.

The patient was digitalized and maintained on a gr. jss dose daily. Nephritin was administered daily and a retention catheter inserted. Sedation with morphine sulfate and atropine sulfate was accomplished. Some improvement and lessening of edema occurred. But on October 24 the patient complained of precordial pain. He was given some sedation. Several hours later he was found dead.

External Examination

The body was that of a white male said to be 75 years of age. The height was 64 inches and the weight was estimated to be 135 to 140 pounds. Only a minor degree of emaciation was present and was that of senility. The head was well shaped. A thin covering of graying brown hair surrounded a bald crown. The left pupil was slightly larger than the right. On the left, the bridge of the nose showed a cicatrix probably representing the irradiation of an epithelioma. False dentures were present in both jaws. The neck and chest were covered by occasional epithelial tumors. The abdomen was scaphoid. No changes of note were found in the upper extremities. On the right knee, a large sized tumor was located. It was probably an epithelioma. The left leg was more edematous than the right and consequently larger. Varicosities were noted about the foot and ankle on the left.

Internal Examination

About 2 to 3 cm. of edematous fat was found overlying the abdomen.

The pericardium showed no gross changes and contained 30 cc. of clear amber fluid.

The heart measured 15 x 12 x 8.5 cm. and weighed 450 gms. The cardiothoracic ratio was 15/21. Marked dilatation of all chambers was present, accompanied by softening of the myocardium which was pallid. Many mural thrombi were contained in the right atrium. The tricuspid valve was compatible with his age. The wall of the right ventricle showed evidence of previous hypertrophy and multiple scarring of coronary occlusive disease. The interventricular septum was thin and necrotic due to a major infarction. Thinness and multiple scarring of the left ventricular wall was noted. Mural thrombi were contained. The mitral curtain was curled upon itself. The anterior descending limb of the left coronary artery was occluded by atherosclerosis and fresh hemorrhage. Atheromatous patches were present throughout the remaining coronary system. The first portion of the aorta was calcified to such an extent that the coronary openings were narrowed. Atheromatous patches and ulcers mottled the abdominal portion especially.

The right pleural sac contained 675 cc. of amber colored fluid and was obliterated by fine adhesions in the region of the apex of the lung.

On the left only a few cubic centimeters of fluid was found. Obliteration of this cavity by adhesions occurred posteriorly.

The right lung weighed 460 gms. A reinfection lesion of tuberculosis was present in the apex. A hemorrhagic infarction was present in the lower portion of the upper lobe. A few small thrombi were found in the pulmonary vessels.

The left lung weighed 350 gms. but showed few changes except obliteration of the interlobar fissure and a few static thrombi.

No esophageal lesions were found. The stomach contained a moderate amount of fluid but showed little or no change.

No intestinal lesions were found.

The liver weighed 1600 gms., being 21 cm. tall. On cross section it proved to be edematous.

Cholesterolosis of the gallbladder was present but no calculi were found.

All ducts allowed free drainage.

Enlarged lymph nodes were noted about the pancreatic head.

The spleen measured 10 x 9 x 5 cm. and weighed 340 gms. The capsule was rusty in color.

There were no lesions of the suprarenal glands.

The right kidney measured 11.5 x 3.5 x 7 cm. and weighed 150 gms. A large solitary cyst measuring 4.5 cm. in diameter was found centrally. Cross section showed the presence of infection.

On the left, the kidney measured 14 x 6 x 6 cm. and weighed 300 gms. Two smaller cysts were present. On cross section infection was noted with hydronephrosis. The right ureter showed no lesions. The left ureter was dilated and contained a calculus approximately 0.5 cm. in diameter about 3 cm. distal to the pelvo-ureteric junction. This portion of the ureter was very congested.

The bladder contained 25 cc. of turbid urine, was congested, and showed many small diverticuli. Projecting into the bladder to the right of the trigone was a soft tumor mass about 1.5 x 2.5 x 2.0 cm. It was found to be infiltrating from its site of origin in the prostate. Upon removal the prostate was enlarged and weighed 110 gms., evidently being nearly entirely replaced by tumor.

The external genitals were compatible with the age of the individual.

Microscopy

Sections of the myocardium show widely separated muscle fibers. This might be due to sectioning but probably is due to edema as is suggested by congested capillaries. Rarely such a vessel is seen in stasis. Edema is also suggested by the poor staining quality of the sarcoplasm and an occasional pyknotic nucleus.

Sections of the liver show good lobular structure with very little fibrous overgrowth. Within the lobules the sinusoids are dilated by edematous fluid. Congestion is noted in the central lobular veins as well as in the interlobular vessels. Occasionally there is round cell infiltration about the biliary radicals.

Sections of the spleen show widespread congestion of the sinusoids to such an extent that normal architecture is lost. Those sinusoids most widely dilated show stasis of their blood content. Distortion is so marked that only the malpighian bodies directly beneath the capsule may be recognized. There is no fibrous overgrowth.

Sections of the kidneys show marked cloudy swelling to the point of disintegration of the tubular epithelium. Quite numerous are the ghosts of glomeruli which now appear as hyaline masses surrounded by inflammatory wandering cells. Occasionally there is a field that is apparently replaced entirely with fibrous connective tissue. The remaining glomeruli are of good size but suggest the possibility of epithelial duplication. Bowman's capsule contains albuminoid debris as do the tubules. Very rarely a small collection of polymorphonuclear neutrophiles are found which might be a beginning abscess. The blood vessels show very little if any change. Toward the apices of the pyramids, congestion is severe with some interstitial hemorrhage. Hyaline casts are numerous in the collecting tubules.

Sections of the prostate show the presence of anaplastic epithelium arranged in some areas to form atypical acini and which are invading the remaining normal stroma. Very hyperchromic and irregularly shaped cells are crowded together in great numbers and thickness to form papillae, and the lining of acini. In other areas, the cells are larger but have the same wild architectural arrangement. In still other areas, masses of anaplastic cells showing no differentiation surround minute corpora amylacia. The stroma about these invading cells show small round cell infiltration and some necrosis. Efferent capillaries occasionally show masses of anaplastic epithelium in their lumina.

Sections of a lymph node show a very loose arrangement that suggests edema, the sinusoids being widely dilated. No anaplastic cells are seen.

Microscopic Diagnosis

Myocardial infarction
Chronic glomerulonephritis
Adenocarcinoma of the prostate (Broder's Grade III or IV) with local metastasis

Anatomical Diagnosis

Diffuse atherosclerosis
Coronary occlusive disease

Multiple myocardial infarctions
Dilatation of the heart
Chronic mitral and aortic valvulitis (rheumatic)
Pleural effusion and adhesions
Cholesterolosis of the gallbladder
Congenital cysts of the kidneys
Hydronephrosis of left kidney (infected)
Nephrolithiasis (left ureter)
Diverticuli of urinary bladder
Carcinoma of the prostate
Senility
Widespread epitheliomata of skin

Cause of Death

Acute myocardial insufficiency due to coronary occlusive disease.

Contributory—congestive heart failure.

SUBJECT INDEX

AUTHOR INDEX